STN:Search report

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(FILE 'HOME' ENTERED AT 14:08:28 ON 27 FEB 2008)

FILE 'MEDLINE, SCISEARCH, CAPLUS, BIOSIS' ENTERED AT 14:08:42 ON 27 FEB 2008

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L1          9307 S PHOSPHOLAMBAN OR PLB
L2             8 S L1 AND S16E
L3             2 DUP REM L2 (6 DUPLICATES REMOVED)
L4       4537704 S HEART OR CARDI? OR CARDIO?
L5         13440 S SERCA?
L6          2182 S L5 AND L1
L7          2182 S L6 AND L5
L8           826 S L7 AND GENE
L9           461 DUP REM L8 (365 DUPLICATES REMOVED)
L10          159 S L9 AND PY<=2000
L11           13 S L10 AND GENE THERAPY
L12           13 FOCUS L11 1-
L13           14 S L10 AND MUTAT?
L14          349 S (CARD? OR HEART) AND MUTAT? AND PHOSPHOLAMBAN
L15          177 DUP REM L14 (172 DUPLICATES REMOVED)
L16            6 S L15 AND GENE THERAPY
                 E CHEIN KENNETH?/AU
                 E CHIEN KENNETH?/AU
L17          609 S E1
L18          401 S IKEDA YASUHIRO?/AU
L19          993 S L17 OR L18
L20           43 S L19 AND L1 AND L4
L21           26 DUP REM L20 (17 DUPLICATES REMOVED)
L22           26 SORT L21 PY
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=> d ti so au ab pi l22 6 7 11

L22 ANSWER 6 OF 26 MEDLINE on STN

TI Chronic suppression of heart-failure progression by a pseudophosphorylated mutant of phospholamban via in vivo cardiac rAAV gene delivery.

SO Nature medicine, (2002 Aug) Vol. 8, No. 8, pp. 864-71. Electronic Publication: 2002-07-22.
Journal code: 9502015. ISSN: 1078-8956.

AU Hoshijima Masahiko; Ikeda Yasuhiro; Iwanaga Yoshitaka; Minamisawa Susumu; Date Moto-o; Gu Yusu; Iwatate Mitsuo; Li Manxiang; Wang Lili; Wilson James M; Wang Yibin; Ross John Jr; Chien Kenneth R

AB The feasibility of gene therapy for cardiomyopathy, heart failure and other chronic cardiac muscle diseases is so far unproven. Here, we developed an in vivo recombinant adeno-associated virus (rAAV) transcoronary delivery system that allows stable, high efficiency and relatively cardiac-selective gene expression. We used rAAV to express a pseudophosphorylated mutant of human phospholamban (PLN), a key regulator of cardiac sarcoplasmic reticulum (SR) Ca(2+) cycling in BIO14.6 cardiomyopathic hamsters. The rAAV/S16EPLN treatment enhanced myocardial SR Ca(2+) uptake and suppressed progressive impairment of left ventricular (LV) systolic function and contractility for 28-30 weeks, thereby protecting cardiac myocytes from cytopathic plasma-membrane disruption. Low LV systolic pressure and deterioration in LV relaxation were also largely prevented by rAAV/S16EPLN treatment. Thus, transcoronary gene transfer of S16EPLN via rAAV vector is a potential therapy for progressive dilated cardiomyopathy and

associated heart failure.

L22 ANSWER 7 OF 26 CAPLUS COPYRIGHT 2008 ACS on STN
TI High efficiency cardiac gene transfer with adeno-associated virus vectors and uses in gene therapy for cardiac diseases
SO U.S. Pat. Appl. Publ., 12 pp.
CODEN: USXXCO
IN Chien, Kenneth R.; Hoshijma, Masahiko; Ross, John; Ikeda, Yasuhiro
AB The present invention discloses methods for the delivery of genes to improve cardiac function including the use of adeno-associated virus (AAV) vectors, isolation of the heart from systemic circulation, and induction of hypothermia/cardiac arrest. The methods result in high-level, long-term expression of reporter genes and enhanced cardiac function in hamster models of heart disease. In particular, the gene expression via AAV vectors is highly restricted to cardiac muscle and maintained long-term, with no sign of myocardial inflammation. Transfer of a gene for a dominant neg. form of phospholamban enhanced the contractility in the heart of hamsters, suppressing heart failure by enhancing the function of sarcoplasmic reticulum calcium ATPase 2.

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2002032167	A1	20020314	US 2001-954571	20010911
CA 2422078	A1	20020321	CA 2001-2422078	20010911
WO 2002022177	A2	20020321	WO 2001-US29103	20010911
WO 2002022177	A3	20021128		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2001091063	A	20020326	AU 2001-91063	20010911
EP 1317289	A2	20030611	EP 2001-971139	20010911
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

L22 ANSWER 11 OF 26 MEDLINE on STN
TI Chronic phospholamban inhibition prevents progressive cardiac dysfunction and pathological remodeling after infarction in rats.
SO The Journal of clinical investigation, (2004 Mar) Vol. 113, No. 5, pp. 727-36.
Journal code: 7802877. ISSN: 0021-9738.
AU Iwanaga Yoshitaka; Hoshijima Masahiko; Gu Yusu; Iwatate Mitsuo; Dieterle Thomas; Ikeda Yasuhiro; Date Moto-o; Chrast Jacqueline; Matsuzaki Masunori; Peterson Kirk L; Chien Kenneth R; Ross John Jr
AB Ablation or inhibition of phospholamban (PLN) has favorable effects in several genetic murine dilated cardiomyopathies, and we showed previously that a pseudophosphorylated form of PLN mutant (S16EPLN) successfully prevented progressive heart failure in cardiomyopathic hamsters. In this study, the effects of PLN inhibition were examined in rats with heart failure after myocardial infarction (MI), a model of acquired disease. S16EPLN was

delivered into failing hearts 5 weeks after MI by transcoronary gene transfer using a recombinant adeno-associated virus (rAAV) vector. In treated (MI-S16EPLN, n = 16) and control (MI-saline, n = 18) groups, infarct sizes were closely matched and the left ventricle was similarly depressed and dilated before gene transfer. At 2 and 6 months after gene transfer, MI-S16EPLN rats showed an increase in left ventricular (LV) ejection fraction and a much smaller rise in LV end-diastolic volume, compared with progressive deterioration of LV size and function in MI-saline rats. Hemodynamic measurements at 6 months showed lower LV end-diastolic pressures, with enhanced LV function (contractility and relaxation), lowered LV mass and myocyte size, and less fibrosis in MI-S16EPLN rats. Thus, PLN inhibition by in vivo rAAV gene transfer is an effective strategy for the chronic treatment of an acquired form of established heart failure.